ADDENDUM TO THE TOXICOLOGICAL PROFILE FOR METHYL MERCAPTAN

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ADDENDUM for Methyl Mercaptan
Supplement to the 1992 Toxicological Profile for Methyl Mercaptan

Background Statement

This addendum to the Toxicological Profile for Methyl Mercaptan supplements the profile released in 1992.

Toxicological profiles are developed in response to the Superfund Amendments and Reauthorization Act (SARA) of 1986, which amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund.) CERCLA further states that the Administrator will "establish and maintain inventory of literature, research, and studies on the health effects of toxic substances" (Title 42, Chapter 103, Subchapter I, § 9604 [i] [1] [B]).

This addendum is a non-peer–reviewed supplement containing scientific data that were published in the open peer-reviewed literature since the release of the toxicological profile in 1992.

Chapter numbers in this addendum coincide with the Toxicological Profile for Methyl Mercaptan (1992). This document should be used with the profile; it does not replace it.

2. HEALTH EFFECTS

2.1 INTRODUCTION

Methyl mercaptan (CH_3SH) is a substance used in manufacturing and other industries. It has a strong odor and is added to natural gas, for example, to help detect leaks. It is also a waste product in certain industries, such as papermaking. As such, its toxicity, combined with production in large amounts, represents a threat to human health. Methyl mercaptan and related substances, such as dimethyl sulfide $(CH_3)_2S$, dimethyl disulfide (CH_3SSCH_3), and hydrogen sulfide (H_2S), are also produced in large concentrations by bacteria in anaerobic sediments and by intestinal microflora. These substances are also present in nontoxic concentrations in wine, cheese, and other foods.

The odor of methyl mercaptan is noticeable at concentrations much lower than those that are hazardous. People can smell methyl mercaptan at about 2 ppb in air, whereas studies in rats and mice show that the concentrations detrimental to health are greater than 100 ppm ([EPA] 2008). Thus, most people can become aware of the presence of hydrogen sulfide and methyl mercaptan at levels significantly below those considered harmful to human health. However, after prolonged exposure, the smell can become less noticeable (Brenneman et al. 2000). When such olfactory fatigue occurs, a person's sense of smell might not provide adequate warning of hazardous air levels.

A question that needs further research is whether methyl mercaptan is genotoxic at concentrations to which humans are often exposed. (See the Genotoxicity section for a discussion on the subject.)

Methyl mercaptan is also known as methanethiol and often abbreviated as MeSH.

2.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

2.2.1 Inhalation Exposure

2.2.1.1 Death

The 1992 Toxicological Profile for Methyl Mercaptan ([ATSDR] 1992) lists a single death after exposure to methyl mercaptan, that of a worker who emptied tanks containing methyl mercaptan (Shults et al. 1970). In a later document, EPA noted the reported death of a 19-year-old exposed to greater than 10,000 ppm of methyl mercaptan for a few minutes ([EPA] 2008). "Death ensued in 45 minutes as a result of respiratory arrest and 'heart failure.' The blood concentration of methyl mercaptan was greater than 2.5 nmol/mL (Syntex Corporation 1979)."

The EPA report also cites a report from Shertzer (2001) of a third death: "A 24-year-old male working in a sodium methyl sulfhydrate factory was found dead. Large quantities of methyl mercaptan were detected in his liver, kidneys, lungs, blood, urine, and in the washout solution of his trachea."

Table 1 lists, for comparison, data included in the Toxicological Profile for Methyl Mercaptan ([ATSDR] 1992).

Table 1. LC_{50} for Sprague-Dawley rats of both sexes acutely exposed for 4-hour periods (Tansy et al. 1981)

	LC_{50} (ppm)
Hydrogen sulfide	444
Methyl mercaptan	675
Dimethyl disulfide	805
Dimethyl sulfide	40,250
Equimolar mixture of methyl mercaptan, dimethyl sulfide, and dimethyl disulfide	550

Respiratory. Researchers who collected air samples from 10 kraft pulp mills found concentrations of 0–20 ppm hydrogen sulfide and 0–15 ppm methyl mercaptan (Kangas et al. (1984). Comparable amounts of dimethyl sulfide with dimethyl disulfide were as high as 1.5 ppm. In a clinical survey of the pulp mill workers, subjective symptoms included increased chronic or recurrent headaches compared to unexposed controls ($p<0.025$). Other effects (not

statistically significant) included decreased mental concentration capacity and nervous system symptoms such as restlessness and lack of vigor.

In its "2008 Interim Acute Exposure Guideline Levels (AEGLs) for Methyl Mercaptan," EPA cites two non-peer–reviewed reports, one on rats ([DuPont] 1992) and one on mice ([SRI] 1996) ([EPA] 2008). Tables 2, 3, and 4, and Figure 1 show data extracted from these documents and summarized by EPA ([EPA] 2008).

TABLE 2. Acute inhalation toxicity in rats exposed to methyl mercaptan

Concentration (ppm)	Duration (hours)	Mortality	Clinical signs	Necropsy findings
250	4	0/2	Ocular and nasal irritation	Pneumonitis in 2 rats; considered coincidental
500	4	0/2	Ocular and nasal irritation, shallow respiration	Focal atelectasis (9 days after treatment in 2 rats)
750	3–3.5	2/2	Comatose a few minutes before death	none
1000	3.17	2/2	Shallow respiration, cyanosis, comatose in 3 hours	none
2000	0.33	2/2	Comatose in 15 minutes	none

Source: ([DuPont] 1992) as cited in ([EPA] 2008)

TABLE 3. Subchronic Inhalation Toxicity in Rats Exposed to Methyl Mercaptan

Concentration	**Duration (ppm)**	**Mortality**	**Clinical Signs**	**Necropsy Findings**
100	6 hours/day x 10 days	0/2	Occasional restlessness	Bronchopneumonia (both rats)
200	6 hours/day x 6 days	1/4	Occasional restlessness, ears red	No effects (2 rats) pneumonia (2 rats, including decedent)
200	6 hours/day x 10 days	1/4	Slight dyspnea and chromodacryorrhea after sixth exposure, slight cyanosis, moist rales (decedent)	Decedent: bronchopneumonia Rat #2: coincidental atelectasis Rat # 3: slight pulmonary congestion and emphysema Rat #4: slight bronchitis and emphysema, coincidental atelectasis

Source: ([DuPont] 1992) as cited in ([EPA] 2008)

TABLE 4. Clinical signs and mortality in Swiss-Webster mice following acute nose-only inhalation exposure to methyl mercaptan for 6 hours.		
Concentration (ppm)	**Clinical Signs**	**Mortality**
0	None	None of 15 mice
114	None	None of 15 mice
258	Shallow breathing and hypoactivity in all mice during the fourth and fifth hours of exposure; appeared normal by day 2	None of 15 mice
512	Shallow breathing in all mice at the third and fourth hours of exposure and hypoactivity during the fifth hour of exposure	Out of 15 mice, 3 female and 2 male mice were found dead on day 2; all surviving mice group appeared normal by day 2.
Source: [SRI] (1996) as cited in ([EPA] 2008)		

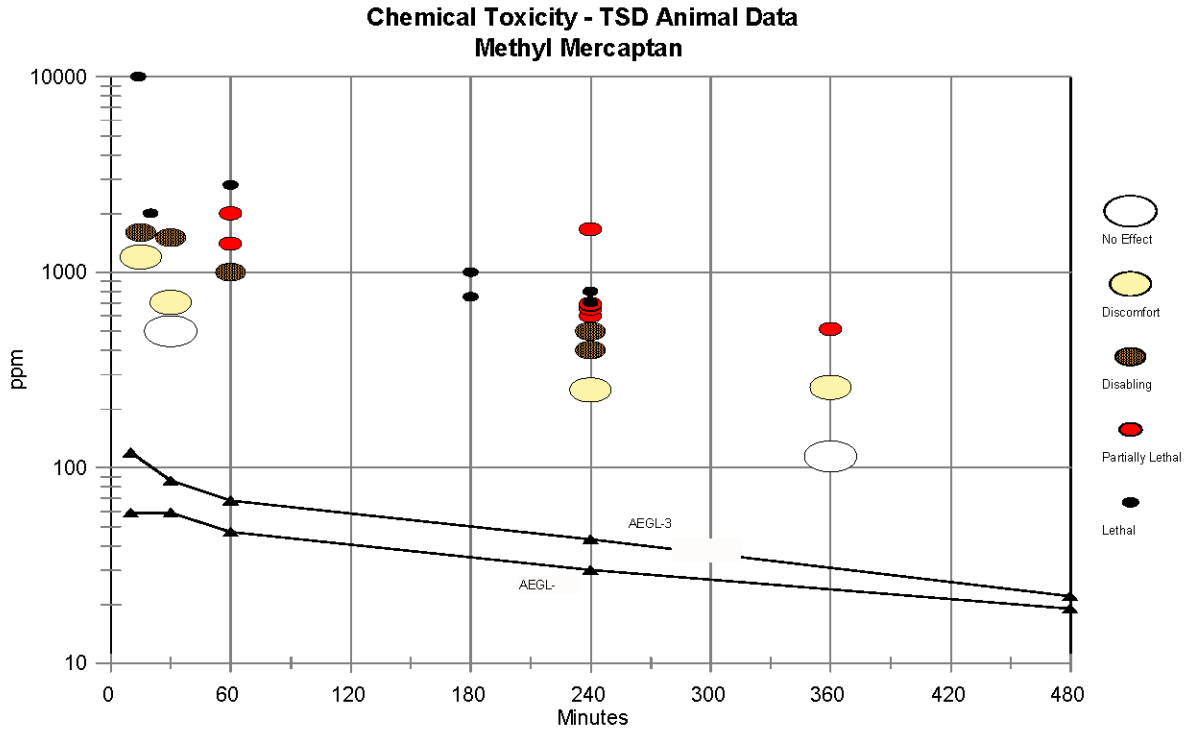

Figure 1. Category plot for chemical toxicity animal data for methyl mercaptan (extracted from ([EPA] 2008)); triangles represent interim acute exposure guideline levels [AEGL])

2.2.1.7 Genotoxic Effects

[SRI] (1996) performed bone marrow micronucleus assays of male and female Swiss-Webster mice following acute nose-only inhalation of methyl mercaptan ([EPA] 2008). A statistically significant increase in micronucleated polychromatic erythrocytes was observed in male mice after exposure to 512 ppm methyl mercaptan for 6 hours. However, exposure to 500 ppm in air can lead to death, rendering the genotoxic effects moot. EPA questioned the biological significance of the findings because of unexpected values in the control group ([EPA] 2008).

Notwithstanding the lack of genotoxicity data for methyl mercaptan, data from hydrogen sulfide studies suggest that methyl mercaptan might be genotoxic in vivo, given the similarity of its toxicity mechanisms with those of hydrogen sulfide. Attene-Ramos et al. (2010) determined that hydrogen sulfide is genotoxic to nontransformed human intestinal epithelial cells at concentrations (found in the intestines) as low as 250 μM. The authors suggest that hydrogen sulfide could be linked to chronic disorders such as ulcerative colitis and colorectal cancer.

Methyl mercaptan, together with hydrogen sulfide and dimethyl sulfide, are the three most common volatile sulfur substances found in the human colon (Suarez et al. 1998). Quite often, the concentration of methyl mercaptan is higher than that of hydrogen sulfide. One study found that the methyl mercaptan concentration exceeded that of hydrogen sulfide in 22% of samples (Suarez et al. 1998).

As discussed below, the colon can quickly detoxify hydrogen sulfide and methyl mercaptan to thiosulfate (Levitt et al. 1999).

2.3 TOXICOKINETICS

The primary source of human exposure to methyl mercaptan is the gas generated in the large intestine. Intestinal microflora can generate concentrations of methyl mercaptan, hydrogen sulfide, and dimethyl sulfide in excess of 1,000 ppm. However, the lining of the colon rapidly detoxifies methyl mercaptan via mechanisms that oxidize methyl mercaptan and hydrogen sulfide to thiosulfate, resulting in no or minimal absorption of methyl mercaptan (Levitt et al.

1999). For example, the oxidation rate of hydrogen sulfide by colonic mucosa is 10,000 times greater than the reported methylation rate (Levitt et al. 1999). Thus, the actual concentrations of these gases expelled in the flatus range from 0.2 ppm to 30 ppm (Furne et al. 2001; Levitt et al. 1999; Suarez et al. 1997).

Human bodies also generate methyl mercaptan in other ways. In the mouth, for example, methyl mercaptan generated around the teeth and gums can contribute to adult periodontitis and inhibit healing surgical wound healing by adversely affecting cell function (Johnson et al. 1992; Lancero et al. 1996; Nakano et al. 2002).

In the large intestine, a detoxification mechanism quickly oxidizes methyl mercaptan (Levitt et al. 1999). This presumably does not happen in the lungs, and inhalation of methyl mercaptan continues to be the primary hazard to human health. Suarez et al. (1998) observed concentrations of 2,000 ppm hydrogen sulfide and 500 ppm methyl mercaptan in gas aspirated from the ceca of rats (Jiang et al. 2001). These are concentrations that would be lethal in less than 1 hour had the exposure occurred in the lungs.

4. PRODUCTION, IMPORT, USE, AND DISPOSAL

4.4 DISPOSAL

Oxidizing agents can be used to control methyl mercaptan and other sulfur gas emissions from sewage sludge. Potassium permanganate and hydrogen peroxide are most effective in reducing methyl mercaptan and dimethyl sulfide emissions, followed by sodium hypochlorite and ferric chloride. Potassium nitrate had no effect on the removal of the sulfur gases (Devai and Delaune 2002).

Methyl mercaptan also can be trapped and oxidized in activated carbon filters by inorganic mechanisms. Activated carbon can remove methyl mercaptan by absorption. By this process, the compound is oxidized to disulfides. Depending on the chemistry of the carbon surface, the disulfides might then be converted to sulfonic acid in the presence of water and active radicals (Bashkova et al. 2002).

Removal of Methyl Mercaptan by Aerobic and Anaerobic Biological Treatment

Thiobacillus thioparus can oxidize hydrogen sulfide, methyl mercaptan, dimethyl sulfide [$(CH_3)_2S$], and dimethyl disulfide (CH_3SSCH_3). The effectiveness of this aerobic bacterium in removing the gases was demonstrated in tests of a pilot-scale peat biofilter treating the exhaust gas from a night soil treatment plant. Average removal ratios with *T. thioparus* inoculated into the biofilter were of 99.8% for hydrogen sulfide, 99.0% for methyl mercaptan, 89.5% for dimethyl sulfide, and 98.1% for dimethyl disulfide. No acclimation period was needed. The pH needs to be controlled to maintain removal efficiency (Cho et al. 1992; Park et al. 1993).

Biological deodorization of dimethyl sulfide was best achieved using an activated carbon fabric in a biofilter. IM1, the probable dominant bacterial strain isolated from the biofilter, degraded dimethyl sulfide, hydrogen sulfide, methyl mercaptan, and dimethyl disulfide (Tiwaree et al. 1992).

A packed bed filter filled with immobilized microorganism beads was studied for removing hydrogen sulfide and methyl mercaptan from wastewater treatment facilities. Optimum pH for the removal of hydrogen sulfide and methyl mercaptan differed markedly. The values were 2-3 and 6-8, respectively. A two-stage biofilter operated in series under different pH values might remove the hydrogen sulfide and methyl mercaptan more effectively (Pinjing et al. 2001).

Ruokojärvi et al. (2001) used similar strategies. The authors connected in series two biotrickling filters with different microbes and operating pH levels to create a two-stage system. The first filter operated at a pH of 2. It removed most of the hydrogen sulfide and some of the methyl mercaptan and dimethyl sulfide. The second filter, at a pH of approximately 6.5, removed the rest of the methyl mercaptan and most of the dimethyl sulfide. The total maximum loads of the whole two-stage biotrickling filter, counted in sulfur amounts, were 1,150 g/m^3/day for hydrogen sulfide, 879 g/m^3/day for dimethyl sulfide, and 66 g/m^3/day for methyl mercaptan treated in a gas mixture. The average removal efficiencies for all gases tested were 99% or higher.

An aerobic enrichment culture grown on a mixture of hydrogen sulfide and methyl mercaptan as sole energy sources was used to remove hydrogen sulfide and methyl mercaptan from a sulfide-

laden spent-sulfidic caustic waste stream. The culture was immobilized in support matrix in a continuous flow, fluidized-bed column bioreactor. Complete oxidation was achieved at sulfate levels below 12 g/L (Conner et al. 2000). *Clostridium methoxybenzovorans* sp. nov., an isolate from an anaerobic methanogenic pilot-scale digester fed with olive oil mill wastewater, was capable of *O*-demethylating a wide range of methoxylated aromatic compounds. Methyl mercaptan and dimethyl sulfide were fermented to acetate (Mechichi et al. 1999b).

Volatile sulfurs such as methyl mercaptan, dimethyl sulfide, and dimethyl disulfide can be removed from wastewater by anaerobic treatment. In one study, biomass originating from an anaerobic wastewater treatment facility treating brewery wastewater converted the three gases to methane and hydrogen sulfide (Sipma et al. (2002). Conversion to methane occurred mainly through respiration by anaerobic bacteria (methanogenesis), as indicated by inhibition of the conversion process with 2-bromoethanesulfonic acid, an inhibitor of bacterial methanogenesis. This process does not address the removal of hydrogen sulfide.

Bioaugmentation is another method for methyl mercaptan odor control and to reduce gaseous emissions from biosolids. This process consists of adding cultured bacteria and nutrients to improve the digestion process. For example, anaerobic biosolids bioaugmented with a commercial product containing selected strains of bacteria from the genera *Bacillus*, *Pseudomonas*, and *Actinomycetes*, organic compounds, and micronutrients, generated 29% more net methane during 8 weeks of operation. As expected, increased methane production removed most of the methyl mercaptan in the bioaugmented digester. The resulting methyl mercaptan concentration was only 37% of the control concentration of nearly 300 ppm(v) (Duran et al. 2006).

Some of the bacteria capable of converting methyl mercaptan to methane gas show sustained growth only on methyl mercaptan and not on related substrates such as methanol, methylamine, or dimethyl sulfide. This was demonstrated using a laboratory-scale anaerobic sludge blanket reactor to which granular sludge from a pulp mill wastewater treatment plant was added. The wastewater methyl mercaptan was degraded at 30°C to hydrogen sulfide, carbon dioxide, and methane. At a volumetric loading rate of 16.5 mmol/liter/day, a bacterial succession was

observed. Methanogenic bacteria related to the genus *Methanolobus* grew initially, but eventually were outcompeted by *Methanomethylovorans hollandica*. Some of the species related to the latter could only show sustained growth on methyl mercaptan (de Bok et al. 2006).

Land application of wastewater biosolids is economical and beneficial to resource recycling. However, this environmentally friendly practice is sometimes restricted because of odor complaints. Methyl mercaptan, dimethyl sulfide, and dimethyl disulfide are among the volatile organic sulfur compounds that contribute to biosolids odor. These odors can be controlled if precautions are taken to store biosolids under anaerobic conditions and proper temperature. Proper temperature can promote the growth of methanogenic bacteria that convert the volatile compounds to methane and carbon dioxide (Chen et al. 2005).

5. POTENTIAL FOR HUMAN EXPOSURE

5.2 RELEASES TO THE ENVIRONMENT

On a global scale, the two primary sources of methyl mercaptan are demethoxylation of lignin byproducts and the amino acid methionine. Lignin, together with cellulose and hemicellulose, is a component of wood, and thus among the most abundant polymers in living ecosystems. Methionine is essential to life, and thus present in all living organisms. The formation of methyl mercaptan from lignin requires the presence of hydrogen sulfide, the formation from methionine does not. The requirement for hydrogen sulfide in the formation of methyl mercaptan from lignin implies the presence of anaerobic conditions.

In anaerobic freshwater sediments, methyl mercaptan formation is a function of the concentrations of sulfide and methyl group-donating compounds. Demethoxylation of syringate (an intermediate microbial metabolite of lignin) is one of the primary sources of methyl mercaptan. The degradation of methyl mercaptan to methane is mediated by a group of obligately methylotrophic methanogens that are phylogenetically related to *M. hollandica* (Lomans et al. 2001b).

Holophaga foetida gen. nov., sp. nov., a gram-negative, obligately anaerobic, rod-shaped bacterium isolated from a black anoxic freshwater mud sample produced dimethyl sulfide and methyl mercaptan during growth on trimethoxybenzoate or syringate. The products originate from the transmethylation of lignin-derived methyl groups and inorganic sulfide (Liesack et al. 1994).

Sporobacterium olearium gen. nov., sp. nov., strain SR1T grew on coronate, methanol, and a number of aromatic compounds as carbon and energy sources. Those compounds included 3,4,5-trimethoxybenzoate (TMB), 3,4,5-trimethoxycinnamate (TMC), syringate, 3,4,5-trimethoxyphenylacetate (TMPA), 3,4,5-trimethoxyphenylpropionate (TMPP), ferulate, sinapate, vanillate, 3,4-dimethoxybenzoate, 2,3-dimethoxybenzoate, gallate, 2,4,6-trihydroxybenzoate (THB), pyrogallol, phloroglucinol, and quercetin. Methyl mercaptan was produced from methoxylated aromatic compounds and methanol (Mechichi et al. 1999a). *Parasporobacterium paucivorans* produced methyl mercaptan and dimethyl sulfide from the methoxy groups of syringate only in the presence of sulfide (Lomans et al. 2001a).

5.3 Environmental Fate

5.3.2 Transformation and Degradation

Microbial aerobic oxidation of methyl mercaptan has been shown to occur by *Thiobacillus* (Subramaniyan et al. 1998; Visscher and Taylor 1993); Kim et al. 1999), *Hyphomicrobium* (Pol et al. 1994), *Pseudomonas* (Honma and Akino 1998), *Methylophaga* (Schäfer 2007), *Marinobacterium* (Fuse et al. 2000), and *Klebsiella* (Seiflein and Lawrence 2001).

The two most common mechanisms of anaerobic oxidation of methyl mercaptan by microbes are sulfate reduction (Eq. 1) and methanogenesis (Eq. 2) (Lomans et al. 2001b).

$$4CH_3SH + 3SO_4^{2-} \rightarrow 4HCO_3^- + 7HS^- + 5H^+ \quad \text{(Eq. 1)}$$

$$4CH_3SH + 3H_2O \rightarrow 3CH_4 + HCO_3^- + 4HS^- + 5H^+ \quad \text{(Eq. 2)}$$

Three thermophilic strains of *Desulfotomaculum* MTS5, TDS2, and SDN4 isolated from a sludge fermentor completely oxidized methyl mercaptan or dimethyl sulfide with sulfate or nitrate as

electron acceptors. The byproducts were carbon dioxide and sulfide or ammonia (Tanimoto and Bak 1994).

5.3.2.2 Water

The *Roseobacter* group of marine bacteria is numerically important in coastal seawater and sediments. Several isolates have been shown to degrade dimethyl sulfide and methyl mercaptan. This group of bacteria can play an important role in the cycling of sulfur compounds in the marine environment. (González et al. 1999).

Aerobic degradation rates for dimethyl sulfide and methyl mercaptan measured under controlled conditions in the lab were 10-fold higher than the maximal anaerobic degrading capabilities. However, under simulated conditions in freshwater sediments the aerobic degradation of dimethyl sulfide and methyl mercaptan was low due to oxygen limitation. Thus, the evidence suggests that in freshwater sediments, conversion to methane is the major mechanism of dimethyl sulfide and methyl mercaptan degradation (Lomans et al. 1999c). These two sulfur compounds can also be oxidized to carbon dioxide by sulfate reducing bacteria when sulfate is present in the sediments. The possibility also exists that the compounds are degraded by a syntrophic association of sulfate or nitrate reducers, and methanogenic bacteria (Lomans et al. 1999b). *M. hollandica* gen. nov., sp. nov., is an example of a methanogen found in freshwater sediments that is able to grow on dimethyl sulfide and methyl mercaptan (Lomans et al. 1999a).

Lomans et al. (2002) studied the general mechanisms for the production and degradation of methyl mercaptan and dimethyl sulfide in anoxic environments. They found that methylation of sulfide was a major mechanism in the generation of methyl mercaptan and dimethyl sulfide. The methyl groups originated from the byproducts of lignin degradation, e.g., syringate. They isolated an anaerobic bacterium that formed methyl mercaptan and dimethyl sulfide with syringate as a methyl group donating compound and sole carbon source. They also isolated a methanogenic bacterium that grew on dimethyl sulfide as the sole carbon source. Methyl mercaptan and dimethyl sulfide produced in freshwater sediments are consumed quickly by methanogenic bacteria, which convert the two gases to methane. When sulfate is present, the two gases are oxidized to carbon dioxide by sulfate-reducing bacteria. A large survey of sediments

slurries of various origin demonstrated that both isolates commonly occur in anaerobic freshwater sediments (Lomans et al. 2002).

5.3.2.3 Soil

Organobromine compounds, including methyl bromide, are produced by a large array of marine organisms (Gribble 2000). Methyl bromide is degraded sequentially to methyl mercaptan and dimethyl sulfide in anaerobic saltmarsh sediments. The two sulfur molecules are in turn converted to methane by methanogenic bacteria (Oremland et al. 1994).

Methyl mercaptan is also a byproduct of the degradation of two oxime carbamate pesticides: oxamyl and methomyl. Iron (Fe[II]) and copper (Cu[I]) can catalyze the inorganic reaction (Strathmann and Stone 2001). The pesticide ethoprop (Mocap) exhibits a similar phenomenon when applied on potato fields, with the subsequent release of n-propyl mercaptan, an odorous compound. These odors were correlated in a survey with a series of symptoms such as headaches, asthma attacks and burning/itching eyes (Ames and Stratton 1991). The significance of these symptoms has been questioned (Greenberg and Campbell 1992).

Studies have shown that rice paddies in China emit hydrogen sulfide, carbonyl sulfide (COS), methyl mercaptan, carbon disulfide (CS_2), dimethyl sulfide, and dimethyl disulfide. Dimethyl sulfide was the predominant sulfur gas emitted. Emissions vary in time and space. Emissions increased with application of organic manure. Diurnal and seasonal variations were influenced by air temperature and the activity of the rice plant. The annual emission of total volatile sulfur gases from a rice paddy in Nanjing ranged from 4.0 mg to 9.5 mg sulfur/m^2/year. Emissions of dimethyl sulfide ranged from 3.1 mg to 6.5 mg sulfur/m^2/year (Yang et al. 1998).

Serratia odorifera, a root-colonizing bacterium that forms symbiotic relationships with many plants, can generate significant amounts of methyl mercaptan and other volatile sulfur compounds (e.g., dimethyl disulfide and dimethyl trisulfide). In laboratory experiments, *S. odorifera* produced 25 $\mu g \cdot h^{-1}$ of methyl mercaptan. These volatile sulfur compound emissions can inhibit the growth of the test plant *Arabidopsis thaliana* (Kai et al. 2010).

Bending andLincoln (1999) measured concentrations of sulfur compounds during decomposition of leaf tissues of *Brassica juncea* (a species of mustard plant) in sandy-loam and clay-loam soils. In both soils, the volatile sulfur-containing compounds carbon disulfide, dimethyl disulfide, dimethyl sulfide, and methyl mercaptan were the dominant headspace components, with maximum concentrations reaching 88, 39, 406, and 992 nmol g^{-1} dry weight leaf incorporated, respectively, in sandy loam and 152, 22, 119, and 473 nmol g^{-1} dry weight leaf added in clay loam.

Methyl mercaptan and carbon disulfide competitively inhibited methane oxidation in two landfill cover soils at concentrations occurring in landfills (Börjesson 2001).

5.4 Levels Monitored in the Environment

5.4.4 Other Environmental Media

Formation of Methyl Mercaptan in Food

Cheddar cheese gets its flavor, in part, from volatile sulfur compounds such as methyl mercaptan, dimethyl disulfide, dimethyl trisulfide, and hydrogen sulfide. To varying degrees, these compounds are produced from sulfur-containing amino acids (i.e., methionine and cysteine) by bacteria such as lactobacilli and lactococci during fermentation (Seefeldt and Weimer 2000); (Bonnarme et al. 2001); (De Angelis et al. 2002); (Arfi et al. 2003); (Cholet et al. 2007); (Psoni et al. 2007).

Probiotic bacteria are added to foods such as yogurt as live cultures. These bacteria generate several volatile sulfur compounds, including hydrogen sulfide, methyl mercaptan, dimethyl disulfide, and dimethyl trisulfide. Probiotic bacteria include *Lactobacillus acidophilus, L. plantarum, L. rhamnosus, L. reuteri, L. delbrueckii,* and *L. lactis* (Sreekumar et al. 2009).

Methyl mercaptan, together with dimethyl disulfide, 3-(methylsulphanyl) propanol, and 3-(methylsulphanyl) propionic acid, is also the product of malolactic fermentation of wine (a process that follows alcoholic fermentation). These compounds add to the aroma produced by the wine. These sulfur compounds are probably produced via catabolism of methionine by lactic acid

bacteria (e.g., *Oenococcus oeni*) present or added to the wine (Pripis-Nicolau et al. 2004). *O. oeni* isolated from wine produced methyl mercaptan, dimethyl disulfide, methionol, and 3-(methylthio) propionic acid when grown on methionine (Vallet et al. 2008).

Cultivation of edible mushrooms (e.g., *Agaricus bisporus*) can also be a source of methyl mercaptan (above the odor threshold) and other sulfur volatile compounds such as hydrogen sulfide and dimethyl sulfide (Noble et al. 2001). These same mushrooms can also remove methyl mercaptan. The odor typical of those who eat garlic, produced by methyl mercaptan and allyl thiols, could be controlled by adding to the diet foods rich in polyphenols. For example, extracts of the mushroom *A. bisporus* have a deodorizing effect on the methyl mercaptan and allyl thiols generated when garlic is eaten. This appears to be accomplished by a reaction of the sulfur compounds with the polyphenols in the fungal extracts (Tamaki et al. 2007).

A representative isolate from *Pseudomonas fluorescens* and three isolates from non-fluorescent *Pseudomonas* recovered from spoiled commercial ground beef produced volatile sulfur compounds, including methyl mercaptan, dimethyl sulfide, dimethyl disulfide, dimethyl trisulfide, and methyl thioacetate, but not hydrogen sulfide (Intarapichet and Bailey 1993).

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

When eaten, certain foods release amounts of methyl mercaptan that people can smell. Persons who eat a raw garlic clove, for example, can release concentrations of a few parts per million in their mouth (Suarez et al. 1999). Eating asparagus produces a mixture of volatile sulfur compounds, including methyl mercaptan, in urine at concentrations up to several thousand times greater than in persons who did not eat asparagus (Waring et al. 1987). People also are regularly exposed to transient amounts of methyl mercaptan in releases of intestinal gas.

Inhalation in occupational settings is probably a significant human exposure scenario for methyl mercaptan.

Pulp mills that use kraft or sulfite processes to bleach paper, for example, can emit hydrogen sulfide, methyl mercaptan, dimethyl sulfide, dimethyl disulfide, and other volatile sulfur

compounds in varying concentrations. Methyl mercaptan and dimethyl sulfide represent more than 80% of total reduced sulfur (TRS) emissions in mill digester blow and relief gases. Hydrogen sulfide and methyl mercaptan comprise more than 75% of TRS emissions in evaporator gases, combined low volume high concentration (LVHC) non-condensable gases (NCGs), recovery furnaces, and tall oil reactor vents. Hydrogen sulfide, methyl mercaptan, and dimethyl sulfide are each present in significant amounts in stripper off-gases (SOGs) and heavy black liquor tank vents ([NCASI] 2002).

In a clinical survey of pulp mill workers, subjective symptoms of those exposed to sulfur compounds included increased chronic or recurrent headaches compared to unexposed controls ($p<0.025$) (Kangas et al. (1984). Other effects (not statistically significant) included decreased mental concentration capacity and nervous system symptoms such as restlessness and lack of vigor.

Conversely, peak ambient concentrations near a community adjacent to a kraft pulp mill site were not detectable (DL = 200 ppb), although sulfur dioxide (as high as 400 ppb), hydrogen sulfide (as high as 1,800 ppb), and carbonyl sulfide (as high as 1,300 ppb) were measured (ATSDR 2007).

Indoor air methyl mercaptan levels in a construction and demolition debris recycling plant were as high as 750 ppb. Butyl mercaptan was as high as 83 ppb, but little or no methyl mercaptan or butyl mercaptan were detected in the adjacent ambient environment (ATSDR 2006). One house had an indoor measurement of 12 ppb methyl mercaptan, but none outdoors. Methyl mercaptan was also found at 1,204 ppb on a school property adjacent to a landfill (ATSDR and PDOH 2010). Some of these ambient concentrations exceeded the 15-minute recommended exposure limit of 500 ppb (0.5 ppm) set by the National Institute for Occupational Safety and Health ([NIOSH] 2010).

7. REGULATIONS AND ADVISORIES

Table 5 lists EPA exposure guidelines for methyl mercaptan [EPA] (2008). National Research Council AEGL-2 acute exposure guideline levels based on one-third reduction of AEGL-3 values also have been added ([NRC] 2013)

Table 5. Extant Standards and Guidelines for Methyl Mercaptan ([EPA] 2008)

Guideline	Exposure duration				
	10 minute	**30 minute**	**1 hour**	**4 hour**	**8 hour**
AEGL-1	NR	NR	NR	NR	NR
AEGL-2	59 ppm	59 ppm	47 ppm	30 ppm	19 ppm
AEGL-2 ([NRC] 2013)	40 ppm	29 ppm	23 ppm	14 ppm	7.3 ppm
AEGL-3	120 ppm	86 ppm	68 ppm	43 ppm	22 ppm
ERPG-1	-	-	0.005 ppm	-	-
ERPG-2	-	-	25 ppm	-	-
ERPG-3	-	-	100 ppm	-	-
NIOSH IDLH	150 ppm				
NIOSH REL					0.5 ppm
OSHA PEL*					10 ppm
ACGIH TLV-TWA					0.5 ppm
OEL (Swedish)					1 ppm
MAK (German)					0.5 ppm
MAC (Dutch)					0.5 ppm

Abbreviations: ACGIH = American Conference of Governmental Industrial Hygienists; AEGL = acute exposure guideline level; ERPG = Emergency Response Planning Guideline; IDLH = immediately dangerous to life or health; MAC = maximum accepted concentration; MAK = maximum workplace concentration; NIOSH = National Institute for Occupational Safety and Health; NR = no recommendation; OEL = occupational exposure limit; PEL = permissible exposure limit; ppm = parts per million; OSHA = Occupational Safety and Health Administration; REL = recommended exposure limit; TLV-TWA = threshold limit value–time-weighted average.

***Note:** "The OSHA Permissible Exposure Limit (PEL) for General Industry" used to be 0.5 ppm, matching the NIOSH recommended exposure limit (REL). The OSHA PEL value for General Industry went back to the old value of 10 ppm as a result of court action ([NIOSH] 2010).

AEGL-1: The airborne concentration (expressed as parts per million or milligrams per cubic meter [ppm or mg/m^3]) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.

AEGL-2: The airborne concentration (expressed as ppm or $mg/m^3$of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.

AEGL-3: The airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death.

Emergency Response Planning Guidelines (ERPGs) ([AIHA] 2014):

ERPG-1: The maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing other than mild, transient adverse health effects or without perceiving a clearly defined objectionable odor, based on the threshold limit value (TLV).

ERPG-2: The maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action (based on repeated-dose animal experiments).

ERPG-3: The maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing or developing life-threatening health effects. Based on 4-hour acute inhalation exposure of 400 ppm in rats (Tansy et al. 1981).

For NIOSH RELs, "**TWA**" indicates a time-weighted average concentration for up to a 10-hour workday during a 40-hour workweek. A short-term exposure limit (STEL) is designated by "**ST**" preceding the value; unless noted otherwise, the STEL is a 15-minute TWA exposure that should not be exceeded at any time during a workday. A ceiling REL is designated by "**C**" preceding the value; unless noted otherwise, the ceiling value should not be exceeded at any time. (See: NIOSH Pocket Guide to Chemical Hazards: Exposure Limits.)

8. REFERENCES

[AIHA] American Industrial Hygiene Association. 2014. Emergency response planning guidelines. Akron, OH. Available at: https://web.archive.org/web/20140918155051/https://www.aiha.org/get-involved/AIHAGuidelineFoundation/EmergencyResponsePlanningGuidelines/Documents/2014%20ERPG%20Values.pdf.

[ATSDR and PDOH] Agency for Toxic Substances and Disease Registry and Pennsylvania Department of Health. 2010. Letter Health Consultation: Imperial landfill site, Imperial, Allegheny County, Pennsylvania. EPA Facility ID: PAD987285624. Atlanta GA. Available at: http://www.atsdr.cdc.gov/hac/pha/ImperialLandfillSite/Imperial_Landfill_Site%20LHC%205-17-2010.pdf.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1992. Toxicological Profile for Methyl Mercaptan. Atlanta GA. Available at: http://www.atsdr.cdc.gov/ToxProfiles/tp139.pdf.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2006. Health Consultation, Exposure Investigation Report: Air Sampling For Sulfur Gases Warren Township, Trumball County, Ohio. Atlanta GA. Available at: http://www.atsdr.cdc.gov/HAC/pha/WarrenRecyclingAirSampling/WarrenRecycling.pdf.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007. Weyerhaeuser Pulp and Paper Mill, Plymouth, North Carolina, EPA Facility ID: CD991278540. Atlanta GA. Available at: http://www.atsdr.cdc.gov/HAC/pha/WeyerhaeuserPulpPaper/WeyerhaeuserPulp+PaperMillHC032207.pdf.

[DuPont] Haskell Laboratory for Toxicology and Industrial Medicine. 1992. Toxicity of methyl mercaptan. Medical Research Project No. MR-287. Available at: Cited in [EPA] 2008.

[EPA] U.S. Environmental Protection Agency. 2008. Interim Acute Exposure Guideline Levels (AEGLs): Methyl Mercaptan (CAS Reg. No. 74-93-1) p. Interim 2: 10/2008. Available at: http://www.epa.gov/oppt/aegl/pubs/methylmercaptan_interim_oct_2008.pdf.

[NCASI] National Council for Air and Stream Improvement. 2002. Compilation of speciated reduced sulfur compound and total reduced sulfur emissions data for kraft mill sources. Available at: http://www.ncasi.org//Publications/Detail.aspx?id=66.

[NIOSH]. 2010. Appendix G: 1989 Air contaminants update project- Exposure limits NOT in effect National Institute for Occupational Safety and Health NIOSH Pocket Guide to Chemical Hazards. Available at: http://www.cdc.gov/niosh/npg/nengapdxg.html.

[NRC] National Research Council. 2013. Acute exposure guideline levels for selected airborne chemicals: Volume 15. The National Academies Press, Washington, DC. Available at: http://www.nap.edu/openbook.php?record_id=18449&page=44.

[SRI] SRI International. 1996. Bone marrow micronucleus assay in male and female Swiss-Webster mice following acute nose-only inhalation exposure to methyl mercaptan. Prepared for Elf Atochem North America, Inc, by SRI International; Menb Park, CA: Jul 9, 1996. (Final Report. SRI Study No. MMO-95). Available at: http://cfpub.epa.gov/ols/catalog/advanced_brief_record.cfm?&FIELD1=SUBJECT&INPUT1=Methyl%20mercaptan&TYPE1=EXACT&LOGIC1=AND&COLL=&SORT_TYPE=MTIC&item_count=7.

Ames RG and Stratton JW. 1991. Acute health effects from community exposure to *N*-propyl mercaptan from an ethoprop (Mocap®)-treated potato field in Siskiyou County, California. Arch Environ Health: Int J 46(4):213-7. Available at: http://dx.doi.org/10.1080/00039896.1991.9937451.

Arfi K, Amárita F, Spinnler H-E, et al. 2003. Catabolism of volatile sulfur compounds precursors by *Brevibacterium linens* and *Geotrichum candidum*, two microorganisms of the cheese ecosystem. J Biotechnol 105(3):245-53. Available at: http://dx.doi.org/10.1016/j.jbiotec.2003.07.003.

Attene-Ramos MS, Nava GM, Muellner MG, et al. 2010. DNA damage and toxicogenomic analyses of hydrogen sulfide in human intestinal epithelial FHs 74 Int cells. Environ Mol Mutagen 51(4):304-14. Available at: http://dx.doi.org/10.1002/em.20546.

Bashkova S, Bagreev A, Bandosz TJ. 2002. Adsorption of methyl mercaptan on activated carbons. Environ Sci Technol 36(12):2777-82. Available at: http://dx.doi.org/10.1021/es011416v.

Bending GD and Lincoln SD. 1999. Characterisation of volatile sulphur-containing compounds produced during decomposition of *Brassica juncea* tissues in soil. Soil Biol Biochem 31(5):695-703. Available at: http://dx.doi.org/10.1016/s0038-0717(98)00163-1.

Bonnarme P, Arfi K, Dury C, et al. 2001. Sulfur compound production by *Geotrichum candidum* from L-methionine: importance of the transamination step. FEMS Microbiol Lett 205(2):247-52. Available at: http://dx.doi.org/10.1111/j.1574-6968.2001.tb10956.x.

Börjesson G. 2001. Inhibition of methane oxidation by volatile sulfur compounds (CH_3SH and CS_2) in landfill cover soils. Waste Manage & Res 19(4):314-9. Available at: http://dx.doi.org/10.1177/0734242x0101900408.

Brenneman KA, Arden James R, Gross EA, et al. 2000. Olfactory neuron loss in adult male CD rats following subchronic inhalation exposure to hydrogen sulfide. Toxicol Pathol 28(2):326-33. Available at: http://dx.doi.org/10.1177/019262330002800213.

Burdack-Freitag A, Mayer F, Breuer K. 2009. Identification of odor-active organic sulfur compounds in gypsum products. CLEAN – Soil, Air, Water 37(6):459-65. Available at: http://dx.doi.org/10.1002/clen.200800224.

Chen Y, Higgins MJ, Maas NA, et al. 2005. Roles of methanogens on volatile organic sulfur compound production in anaerobically digested wastewater biosolids. Water Sci Technol 52(1-2):67-72. Available at: http://www.iwaponline.com/wst/05201/wst052010067.htm.

Cho K-S, Hirai M, Shoda M. 1992. Enhanced removal efficiency of malodorous gases in a pilot-scale peat biofilter inoculated with *Thiobacillus thioparus* DW44. J Ferment Bioeng 73(1):46-50. Available at: http://dx.doi.org/10.1016/0922-338x(92)90230-r.

Cholet O, Hénaut A, Bonnarme P. 2007. Transcriptional analysis of L-methionine catabolism in *Brevibacterium linens* ATCC9175. Appl Microbiol Biotechnol 74(6):1320-32. Available at: http://dx.doi.org/10.1007/s00253-006-0772-0.

Conner J, Beitle R, Duncan K, et al. 2000. Biotreatment of refinery spent-sulfidic caustic using an enrichment culture immobilized in a novel support matrix. Appl Biochem Biotechnol 84-86(1):707-19. Available at: http://dx.doi.org/10.1385/ABAB:84-86:1-9:707.

De Angelis M, Curtin ÁC, McSweeney PLH, et al. 2002. *Lactobacillus reuteri* DSM 20016: purification and characterization of a cystathionine γ-lyase and use as adjunct starter in cheesemaking. J Dairy Res 69(02):255-67. Available at: http://dx.doi.org/10.1017/S0022029902005514.

de Bok FAM, van Leerdam RC, Lomans BP, et al. 2006. Degradation of methanethiol by methylotrophic methanogenic archaea in a lab-scale upflow anaerobic sludge blanket reactor. Appl Environ Microbiol 72(12):7540-7. Available at: http://dx.doi.org/10.1128/aem.01133-06.

Devai I and Delaune RD. 2002. Effectiveness of selected chemicals for controlling emission of malodorous sulfur gases in sewage sludge. Environ Technol 23(3):319-29. Available at: http://www.ingentaconnect.com/content/tandf/envt/2002/00000023/00000003/art00009.

Duran M, Tepe N, Yurtsever D, et al. 2006. Bioaugmenting anaerobic digestion of biosolids with selected strains of *Bacillus*, *Pseudomonas*, and *Actinomycetes* species for increased methanogenesis and odor control. Appl Microbiol Biotechnol 73(4):960-6. Available at: http://dx.doi.org/10.1007/s00253-006-0548-6.

Furne J, Springfield J, Koenig T, et al. 2001. Oxidation of hydrogen sulfide and methanethiol to thiosulfate by rat tissues: a specialized function of the colonic mucosa. Biochem Pharmacol 62(2):255-9. Available at: http://dx.doi.org/10.1016/s0006-2952(01)00657-8.

Fuse H, Takimura O, Murakami K, et al. 2000. Utilization of dimethyl sulfide as a sulfur source with the aid of light by *Marinobacterium sp.* strain DMS-S1. Appl Environ Microbiol 66(12):5527-32. Available at: http://dx.doi.org/10.1128/aem.66.12.5527-5532.2000.

González JM, Kiene RP, Moran MA. 1999. Transformation of sulfur compounds by an abundant lineage of marine bacteria in the α-subclass of the Class *Proteobacteria*. Appl Environ Microbiol 65(9):3810-9. Available at: http://aem.asm.org/content/65/9/3810.abstract.

Greenberg G and Campbell D. 1992. Letters to the Editor: Acute health effects from community exposure to *n*-propyl mercaptan from an ethoprop (Mocap)-treated potato field in Siskiyou County, California. Arch Environ Health: Int J 47(4):318. Available at: http://dx.doi.org/10.1080/00039896.1992.9938369.

Gribble GW. 2000. The natural production of organobromine compounds. Environ Sci Pollut Res 7(1):37-49. Available at: http://dx.doi.org/10.1065/espr199910.002.

Honma T and Akino T. 1998. Isolation and characterization of a hydrogen sulfide-removing bacterium, *Pseudomonas sp.* Strain DO-1. Biosci Biotechnol Biochem 62(9):1684-7. Available at: http://dx.doi.org/10.1271/bbb.62.1684.

Intarapichet K and Bailey M. 1993. Volatile compounds produced by meat *Pseudomonas* grown on beef at refrigeration temperatures. ASEAN Food J 8(1):14–21.

Jiang T, Suarez FL, Levitt MD, et al. 2001. Gasproduction by feces of infants. J Pediatr Gastroenterol Nutr 32(5):534-41. Available at: http://journals.lww.com/jpgn/Fulltext/2001/05000/Gas_Production_by_Feces_of_Infants.9.aspx.

Johnson PW, Ng W, Tonzetich J. 1992. Modulation of human gingival fibroblast cell metabolism by methyl mercaptan. J Periodontal Res 27(5):476-83. Available at: http://dx.doi.org/10.1111/j.1600-0765.1992.tb01820.x.

Kai M, Crespo E, Cristescu S, et al. 2010. *Serratia odorifera*: Analysis of volatile emission and biological impact of volatile compounds on *Arabidopsis thaliana*. Appl Microbiol Biotechnol 88(4):965-76. Available at: http://dx.doi.org/10.1007/s00253-010-2810-1.

Kangas J, Jäppinen P, Savolainen H. 1984. Exposure to hydrogen sulfide, mercaptans and sulfur dioxide in pulp industry. Am Ind Hyg Assoc J 45(12):787-90. Available at: http://dx.doi.org/10.1080/15298668491400647.

Lancero H, Niu J, Johnson PW. 1996. Exposure of periodontal ligament cells to methyl mercaptan reduces intracellular pH and inhibits cell migration. J Dent Res 75(12):1994-2002. Available at: http://dx.doi.org/10.1177/00220345960750121201.

Levitt MD, Furne J, Springfield J, et al. 1999. Detoxification of hydrogen sulfide and methanethiol in the cecal mucosa. J Clin Invest 104(8):1107-14. Available at: http://dx.doi.org/10.1172/jci7712.

Liesack W, Bak F, Kreft J-U, et al. 1994. *Holophaga foetida gen. nov., sp. nov.*, a new, homoacetogenic bacterium degrading methoxylated aromatic compounds. Arch Microbiol 162(1):85-90. Available at: http://dx.doi.org/10.1007/BF00264378.

Lomans BP, Leijdekkers P, Wesselink J-J, et al. 2001a. Obligate sulfide-dependent degradation of methoxylated aromatic compounds and formation of methanethiol and dimethyl sulfide by a freshwater sediment isolate, *Parasporobacterium paucivorans gen. nov., sp. nov.* Appl Environ Microbiol 67(9):4017-23. Available at: http://dx.doi.org/10.1128/aem.67.9.4017-4023.2001.

Lomans BP, Luderer R, Steenbakkers P, et al. 2001b. Microbial populations involved in cycling of dimethyl sulfide and methanethiol in freshwater sediments. Appl Environ Microbiol 67(3):1044-51. Available at: http://dx.doi.org/10.1128/aem.67.3.1044-1051.2001.

Lomans BP, Maas R, Luderer R, et al. 1999a. Isolation and characterization of *Methanomethylovorans hollandica gen. nov., sp. nov.*, isolated from freshwater sediment, a methylotrophic methanogen able to grow on dimethyl sulfide and methanethiol. Appl Environ Microbiol 65(8):3641-50. Available at: http://aem.asm.org/content/65/8/3641.abstract.

Lomans BP, Op den Camp HJM, Pol A, et al. 1999b. Role of methanogens and other bacteria in degradation of dimethyl sulfide and methanethiol in anoxic freshwater sediments. Appl Environ Microbiol 65(5):2116-21. Available at: http://aem.asm.org/content/65/5/2116.abstract.

Lomans BP, Op den Camp HJM, Pol A, et al. 1999c. Anaerobic versus aerobic degradation of dimethyl sulfide and methanethiol in anoxic freshwater sediments. Appl Environ Microbiol 65(2):438-43. Available at: http://aem.asm.org/content/65/2/438.abstract.

Lomans BP, Pol A, Op den Camp HJM. 2002. Microbial cycling of volatile organic sulfur compounds in anoxic environments. Water Sci Technol 45(10):55-60. Available at: http://www.iwaponline.com/wst/04510/wst045100055.htm.

Mechichi T, Labat M, Garcia J-L, et al. 1999a. *Sporobacterium olearium gen. nov., sp. nov.*, a new methanethiol-producing bacterium that degrades aromatic compounds, isolated from an olive mill wastewater treatment digester. Int J Syst Evol Micro 49(4):1741-8. Available at: http://dx.doi.org/10.1099/00207713-49-4-1741.

Mechichi T, Labat M, Patel BKC, et al. 1999b. *Clostridium methoxybenzovorans sp. nov.*, a new aromatic *O*-demethylating homoacetogen from an olive mill wastewater treatment digester. Int J Syst Bacteriol 49(3):1201-9. Available at: http://dx.doi.org/10.1099/00207713-49-3-1201.

Nakano Y, Yoshimura M, Koga T. 2002. Methyl mercaptan production by periodontal bacteria. Int Dent J 52 Suppl 3:217-20. Available at: http://ukpmc.ac.uk/abstract/MED/12090456.

Noble R, Hobbs PJ, Dobrovin-Pennington A, et al. 2001. Olfactory response to mushroom composting emissions as a function of chemical concentration. J Environ Qual 30(3):760. Available at: http://dx.doi.org/10.2134/jeq2001.303760x.

Oremland RS, Miller LG, Strohmaier FE. 1994. Degradation of methyl bromide in anaerobic sediments. Environ Sci Technol 28(3):514-20. Available at: http://dx.doi.org/10.1021/es00052a026.

Park S-J, Cho K-S, Hirai M, et al. 1993. Removability of malodorous gases from a night soil treatment plant by a pilot-scale peat biofilter inoculated with *Thiobacillus thioparus* DW44. J Ferment Bioeng 76(1):55-9. Available at: http://dx.doi.org/10.1016/0922-338x(93)90053-b.

Pinjing H, Liming S, Zhiwen Y, et al. 2001. Removal of hydrogen sulfide and methyl mercaptan by a packed tower with immobilized micro-organism beads. Water Sci Technol 44(9):327-33. Available at: http://www.iwaponline.com/wst/04409/wst044090327.htm.

Pol A, Op den Camp HJM, Mees SGM, et al. 1994. Isolation of a dimethylsulfide-utilizing *Hyphomicrobium* species and its application in biofiltration of polluted air. Biodegradation 5(2):105-12. Available at: http://dx.doi.org/10.1007/BF00700635.

Pripis-Nicolau L, de Revel G, Bertrand A, et al. 2004. Methionine catabolism and production of volatile sulphur compounds by *Œnococcus œni*. J Appl Microbiol 96(5):1176-84. Available at: http://dx.doi.org/10.1111/j.1365-2672.2004.02257.x.

Psoni L, Kotzamanidis C, Yiangou M, et al. 2007. Genotypic and phenotypic diversity of *Lactococcus lactis* isolates from Batzos, a Greek PDO raw goat milk cheese. Int J Food Microbiol 114(2):211-20. Available at: http://dx.doi.org/10.1016/j.ijfoodmicro.2006.09.020.

Ruokojärvi A, Ruuskanen J, Martikainen PJ, et al. 2001. Oxidation of gas mixtures containing dimethyl sulfide, hydrogen sulfide, and methanethiol using a two-stage biotrickling filter. J Air Waste Manag Assoc 51(1):11-6. Available at: http://dx.doi.org/10.1080/10473289.2001.10464260.

Schäfer H. 2007. Isolation of *Methylophaga spp.* from marine dimethylsulfide-degrading enrichment cultures and identification of polypeptides induced during growth on dimethylsulfide. Appl Environ Microbiol 73(8):2580-91. Available at: http://dx.doi.org/10.1128/aem.02074-06.

Seefeldt KE and Weimer BC. 2000. Diversity of sulfur compound production in lactic acid bacteria. J Dairy Sci 83(12):2740-6. Available at: http://dx.doi.org/10.3168/jds.S0022-0302(00)75168-X.

Seiflein TA and Lawrence JG. 2001. Methionine-to-cysteine recycling in *Klebsiella aerogenes*. J Bacteriol 183(1):336-46. Available at: http://dx.doi.org/10.1128/jb.183.1.336-346.2001.

Shertzer H. 2001. Organic sulfur compounds. In: Bingham E and Cohrssen B and Powell CH, eds. Patty's Toxicology: Glycols and Glycol/Ethers/Synthetic Polymers/Organic Sulfur Compounds/Organic Phosphates. 5th ed. New York: John Wiley & Sons. p. 681–765.

Shults W, Fountain EN, C. LE. 1970. Methanethiol poisoning: Irreversible coma and hemolytic anemia following inhalation. JAMA - J Am Med Assoc 211(13):2153-4. Available at: http://dx.doi.org/10.1001/jama.1970.03170130049013.

Sipma J, van Bree R, Janssen AJH, et al. 2002. Degradation of methanethiol in a continuously operated upflow anaerobic sludge-blanket reactor. Water Environ Res 74(3):264-71. Available at: http://dx.doi.org/10.2175/106143002X139992.

Sreekumar R, Al-Attabi Z, Deeth HC, et al. 2009. Volatile sulfur compounds produced by probiotic bacteria in the presence of cysteine or methionine. Lett Appl Microbiol 48(6):777-82. Available at: http://dx.doi.org/10.1111/j.1472-765X.2009.02610.x.

Strathmann TJ and Stone AT. 2001. Reduction of the carbamate pesticides oxamyl and methomyl by dissolved Fe^{II} and Cu^{I}. Environ Sci Technol 35(12):2461-9. Available at: http://dx.doi.org/10.1021/es001824j.

Suarez F, Furne J, Springfield J, et al. 1997. Insights into human colonic physiology obtained from the study of flatus composition. Am J Physiol Gastrointest Liver Physiol 272(5):G1028-G33. Available at: http://ajpgi.physiology.org/content/272/5/G1028.

Suarez F, Springfield J, Furne J, et al. 1999. Differentiation of mouth versus gut as site of origin of odoriferous breath gases after garlic ingestion. Am J Physiol Gastrointest Liver Physiol 276(2):G425-G30. Available at: http://ajpgi.physiology.org/content/276/2/G425.

Suarez F, Springfield J, Levitt MD. 1998. Identification of gases responsible for the odour of human flatus and evaluation of a device purported to reduce this odour. Gut 43(1):100-4. Available at: http://dx.doi.org/10.1136/gut.43.1.100.

Subramaniyan A, Kolhatkar R, Sublette K, et al. 1998. Microbial oxidation of mixtures of methylmercaptan and hydrogen sulfide. Appl Biochem Biotechnol 70-72(1):995-1005. Available at: http://dx.doi.org/10.1007/BF02920209.

Syntex Corporation. 1979. Unpublished accident report. TSCA Section 8E submission, TSCAT Accession No. 12079, Fiche No. 0000032-0. Available at: Cited in EPA (2008).

Tamaki K, Tamaki T, Yamazaki T. 2007. Studies on the deodorization by mushroom (*Agaricus bisporus*) extract of garlic extract-induced oral malodor. J Nutr Sci Vitaminol (Tokyo) 53(3):277-86. Available at: http://dx.doi.org/10.3177/jnsv.53.277.

Tanimoto Y and Bak F. 1994. Anaerobic degradation of methylmercaptan and dimethyl sulfide by newly isolated thermophilic sulfate-reducing bacteria. Appl Environ Microbiol 60(7):2450-5. Available at: http://aem.asm.org/content/60/7/2450.abstract.

Tansy MF, Kendall FM, Fantasia J, et al. 1981. Acute and subchronic toxicity studies of rats exposed to vapors of methyl mercaptan and other reduced-sulfur compounds. J Toxicol Environ Health 8(1-2):71-88. Available at: http://dx.doi.org/10.1080/15287398109530051.

Tiwaree R, Cho K-s, Hirai M, et al. 1992. Biological deodorization of dimethyl sulfide using different fabrics as the carriers of microorganisms. Appl Biochem Biotechnol 32(1):135-48. Available at: http://dx.doi.org/10.1007/BF02922154.

Vallet A, Lucas P, Lonvaud-Funel A, et al. 2008. Pathways that produce volatile sulphur compounds from methionine in *Oenococcus oeni*. J Appl Microbiol 104(6):1833-40. Available at: http://dx.doi.org/10.1111/j.1365-2672.2007.03713.x.

Visscher PT and Taylor BF. 1993. Aerobic and anaerobic degradation of a range of alkyl sulfides by a denitrifying marine bacterium. Appl Environ Microbiol 59(12):4083-9. Available at: http://aem.asm.org/content/59/12/4083.abstract.

Waring RH, Mitchell SC, Fenwick GR. 1987. The chemical nature of the urinary odour produced by man after asparagus ingestion. Xenobiotica 17(11):1363-71. Available at: http://dx.doi.org/doi:10.3109/00498258709047166.

Yang Z, Kong L, Zhang J, et al. 1998. Emission of biogenic sulfur gases from Chinese rice paddies. Sci Total Environ 224(1–3):1-8. Available at: http://dx.doi.org/10.1016/s0048-9697(98)00207-1.

Appendix A

Microbial Considerations in the Generation of Volatile Sulfur Substances in Drywall

(This discussion assumes the presence of sufficient moisture and nutrients to stimulate microbial growth.)

Methyl mercaptan is one of the many volatile sulfur compounds generated in drywall (Burdack-Freitag et al. 2009).

Bacterially-mediated processes that could play a role in the generation of volatile sulfur substances in drywall include the following.

1. Drywall is largely made of calcium sulfate ($CaSO_4$).
    a. Most of the gaseous sulfur substances emitted from drywall should be in the form of hydrogen sulfide (H_2S), if the origin of the malodors is caused by biofouling (from the reduction of sulfate to hydrogen sulfide by anaerobic respiration.)
        i. $2[H] + SO_4^{2-} \rightarrow H_2S + H_2O$
2. Reports of pyrite (FeS_2, fool's gold) in drywall.
    a. Pyrite is biodegradable. Acidophilic bacteria can solubilize and oxidize pyrite to ferric ions and sulfuric acid.
    b. The production of sulfuric acid can in turn volatilize any hydrogen sulfide present in the drywall.
    c. Please note that the bacteria that grow on pyrite, also grow on hydrogen sulfide and other volatile sulfur compounds, e.g., methyl mercaptan
3. Reports of strontium sulfide (SrS) in drywall.
    a. Strontium sulfide is slightly soluble in water, but highly soluble in acid.
    b. Acidification owing to microbial fouling can lead to volatilization of H_2S.
        i. $SrS\ (s) + 2H^+\ (aq.) \rightarrow Sr^{2+}\ (aq.) + H_2S\ (g)$
4. Reports of elemental sulfur in drywall.
    a. Under anaerobic conditions elemental sulfur is reduced to hydrogen sulfide.
        i. $2[H] + S^0 \rightarrow H_2S$
    b. Under aerobic conditions elemental sulfur is oxidized to sulfuric acid.
        i. $2\ S + 3\ O_2 + 2\ H_2O \rightarrow 2\ H_2SO_4$
    c. Elemental sulfur can also undergo disproportionation to sulfate and hydrogen sulfide.
        i. $4S^0 + 4H_2O \rightarrow SO_4^{2-} + 3H_2S + 2H^+$
5. Reports of organic matter (e.g., paper [cellulose, hemicellulose, and lignin) or starch in drywall.
    a. Polymers plus acid pH (from the microbial production of organic acids) favor the growth of molds.
    b. Anaerobic degradation of lignin in the presence of hydrogen sulfide leads to the production of methyl mercaptan and dimethyl sulfide.
    c. Anaerobic conditions favor the growth of sulfate-reducing bacteria, leading to the production of hydrogen sulfide.